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         May 15
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                 Simultaneous left and right truncation added to WSCA
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                 RAPRA enhanced with new search field, simultaneous left and
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                 Simultaneous left and right truncation added to CBNB
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                 PASCAL enhanced with additional data
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         Jun 20
                 2003 edition of the FSTA Thesaurus is now available
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         Jul 16
                 Data from 1960-1976 added to RDISCLOSURE
                 Identification of STN records implemented
NEWS 26
         Jul 21
NEWS 27
         Jul 21
                 Polymer class term count added to REGISTRY
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         Jul 22
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                 Right Truncation available
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                 Field Availability (/FA) field enhanced in BEILSTEIN
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         AUG 18
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        AUG 18
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- 2002:56228 AGRICOLA AN
- DN IND23283389
- TI Partial redistribution of the Autographa californica nucleopolyhedrovirus chitinase in virus-infected cells accompanies mutation of the carboxy-terminal KDEL ER-retention motif.
- ΑU Saville, G.P.; Thomas, C.J.; Possee, R.D.; King, L.A.
- ΑV DNAL (QR360.A1J6)
- SO The Journal of general virology, Mar 2002. Vol. 83, No. pt.3. p. 685-694 Publisher: Reading : Society for General Microbiology. CODEN: JGVIAY; ISSN: 0022-1317
- NTEIncludes references
- CY England; United Kingdom
- DT Article
- FS Non-U.S. Imprint other than FAO
- LAEnglish
- AB During virus infection of insect cells, the Autographa californica nucleopolyhedrovirus chitinase is localized primarily within the endoplasmic reticulum (ER), which is consistent with the presence of a carboxy-terminal ER retention motif (KDEL). Release of chitinase into the extracellular medium appears to be concomitant with terminal cell lysis, rather than by active secretion. In this study, we have shown that mutation of the KDEL motif induces a partial redistribution of the chitinase at both early and late times post-infection. Deletion of the KDEL motif or substitution with glycine residues allowed chitinase to move through the secretory pathway, accumulating to detectable levels in the extracellular medium by 24 h post-infection; more than 48 h prior to cell lysis. Deletion of the KDEL motif did not compromise enzyme activity, with the modified enzyme exhibiting characteristic endo- and exo-chitinolytic activity. Trichoplusia ni larvae infected with the modified virus were found to liquefy approximately 24 h earlier than larvae infected with a control virus in which the chitinase KDEL motif had not been deleted.
- L5 ANSWER 2 OF 9 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
- AN 2001:334317 BIOSIS
- DN PREV200100334317
- TI The KDEL receptor mediates a retrieval mechanism that contributes to quality control at the endoplasmic reticulum.
- ΑU Yamamoto, Katsushi; Fujii, Rika; Toyofuku, Yukiko; Saito, Takashi; Koseki, Haruhiko; Hsu, Victor W.; Aoe, Tomohiko (1)
- CS (1) Department of Molecular Embryology, Chiba University Graduate School of Medicine, Chiba, 260-8670: taoe@med.m.chiba-u.ac.jp Japan
- SO EMBO (European Molecular Biology Organization) Journal, (June 15, 2001) Vol. 20, No. 12, pp. 3082-3091. print. ISSN: 0261-4189.
- DTArticle
- LAEnglish
- English SL
- AB Newly synthesized proteins in the endoplasmic reticulum (ER) must fold and assemble correctly before being transported to their final cellular destination. While some misfolded or partially assembled proteins have been shown to exit the ER, they fail to escape the early secretory system entirely, because they are retrieved from post-ER compartments to the ER. We elucidate a mechanistic basis for this retrieval and characterize its contribution to ER quality control by studying the fate of the unassembled T-cell antigen receptor (TCR) alpha chain. While the steady-state distribution of TCRalpha is in the ER, inhibition of retrograde transport by COPI induces the accumulation of TCRalpha in post-ER compartments, suggesting that TCRalpha is cycling between the ER and post-ER

compartments. TCRalpha associates with BiP, a KDEL protein. Disruption of the ligand-binding function of the KDEL receptor releases TCRalpha from the early secretory system to the cell surface, so that TCRalpha is no longer subject to ER degradation. Thus, our findings suggest that retrieval by the KDEL receptor contributes to mechanisms by which the ER monitors newly synthesized proteins for their proper disposal.

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AN 1989:91574 BIOSIS

DN BA87:45710

- TI EVIDENCE THAT LUMINAL ER PROTEINS ARE SORTED FROM SECRETED PROTEINS IN A POST-ER COMPARTMENT.
- AU PELHAM H R B
- CS MRC LAB. MOLECULAR BIOL., HILLS RD., CAMBRIDGE CB2 2QH, UK.
- SO EMBO (EUR MOL BIOL ORGAN) J, (1988) 7 (4), 913-918.

CODEN: EMJODG. ISSN: 0261-4189.

- FS BA; OLD
- LA English
- Several soluble proteins that reside in the lumen of the ER contain a AΒ specific C-terminal sequence (KDEL) which prevents their secretion. This sequence may be recognized by a receptor that either immobilizes the proteins in the ER, or sorts them from other proteins at a later point in the secretory pathway and returns them to their normal location. To distinguish these possibilites, I have attached an ER retention signal to the lysosomal protein cathepsin D. The oligosaccharide side chains of this protein are normally modified sequentially by two enzymes to form mannose-6-phosphate residues; these enzymes do not act in the ER, but are thought to be located in separate compartments within (or near) the Golgi apparatus. Cathepsin D bearing the ER signal accumulates within the ER, but continues to be modified by the first of the mannose-6-phosphate forming enzymes. Modification is strongly temperature-dependent, which is also a feature of ER-to-Golgi transport. These results support the idea that luminal ER proteins are continuously retrieved from a post-ER compartment, and that this compartment contains N-acetylglucosaminyl-1-phosphotransferase activity.
- L5 ANSWER 4 OF 9 CANCERLIT on STN
- AN 91015337 CANCERLIT
- DN 91015337 PubMed ID: 2120591
- TI Secretion of immunoglobulin M assembly intermediates in the presence of reducing agents.
- AU Alberini C M; Bet P; Milstein C; Sitia R
- CS Istituto di Chimica, Facolta di Medicina, Universita degli Studi di Brescia, Italy.
- SO NATURE, (1990 Oct 4) 347 (6292) 485-7. Journal code: 0410462. ISSN: 0028-0836.
- CY ENGLAND: United Kingdom
- DT Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS MEDLINE; Priority Journals
- OS MEDLINE 91015337
- EM 199011
- ED Entered STN: 19941107 Last Updated on STN: 19970509
- AB There are several demonstrations that misfolded or unassembled proteins are not transported along the secretory pathway, but are retained intracellularly, generally in the endoplasmic reticulum. For instance, B lymphocytes synthesize but do not secrete IgM, and only the polymeric form of IgM is secreted by plasma cells. The C-terminal cysteine of the mu heavy chain of secreted IgM (residue 575) is involved in the intracellular retention of unpolymerized IgM subunits. Here we report that the addition of reducing agents to the culture medium, at concentrations which do not affect cell viability, terminal glycosylation, or retention of proteins in

the endoplasmic reticulum through the KDEL mechanism, induces secretion of IgM assembly intermediates by both B and plasma cells. Free joining (J) chains, which are not normally secreted by plasma cells unless as part of IgM or IgA, are also secreted in the presence of reducing agents. We propose a role for free thiol groups in preventing the unhindered transport of proteins through the secretory pathway. Under the scheme, assembly intermediates interact through their thiol groups between themselves and/or with unknown proteins of the endoplasmic reticulum. Such interactions may be prevented by altering the intracellular redox potential or by site-directed mutagenesis of the relevant cysteine residue(s).

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     ANSWER 5 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
     2003:402183 CAPLUS
AN
DN
     138:400516
TΙ
     Manufacture of antibodies in plant cells as fusion proteins with
     elastin-like peptides
     Scheller, Juergen; Conrad, Udo; Leps, Michael
IN
PA
     IPK- Institut Fuer Pflanzengenetik Und Kulturpflanzen Forschung, Germany
SO
     PCT Int. Appl., 62 pp.
     CODEN: PIXXD2
DT
     Patent
LA
     German
FAN.CNT 1
     PATENT NO.
                     KIND DATE
                                          APPLICATION NO. DATE
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PΙ
     WO 2003041493
                     A1 20030522
                                         WO 2002-EP12773 20021114
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             PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,
             NE, SN, TD, TG
     DE 10155862
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                      A1
                           20030528
PRAI DE 2001-10155862 A
                           20011114
     A method of manufg. antibodies, esp. single-chain antibodies, in increased
     yields in plant cells as fusion proteins with elastin-like peptides by
     expression of the corresponding gene is described. Yields of antibody are
     greatly increased when they are manufd. as these fusion proteins compared
     to without them and the protein can be rapidly purified by pptn. The
     fusion protein may contain several repeats of the elastin-like peptide, a
     signal peptide to direct secretion and a KDEL peptide
     for retention in the endoplasmic reticulum. The gene may be expressed in
     seed using a a strong promoter such as the cauliflower mosaic virus 35S
     promoter. Yields of antibody from seed of transgenic tobacco reached 17%
     of total protein.
RE.CNT 7
              THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
```

L5 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN

AN 2000:98760 CAPLUS

DN 132:133894

TI Inhibition of KDEL receptor-mediated return of heat shock protein complexes to the endoplasmic reticulum and their adjuvant use

ALL CITATIONS AVAILABLE IN THE RE FORMAT

IN Rothman, James E.; Mayhew, Mark; Hoe, Mee H.

PA Sloan-Kettering Institute for Cancer Research, USA

SO PCT Int. Appl., 87 pp. CODEN: PIXXD2

DT Patent

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English
LA
FAN.CNT 1
                      KIND DATE
                                            APPLICATION NO. DATE
     PATENT NO.
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                            _____
                                           WO 1999-US17147 19990728
                            20000210
     WO 2000006729
PΤ
                      A1
         W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ,
             DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,
             JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,
             MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
             TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD,
             RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
             ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,
             CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
                             20001212
                                           US 1998-124671
                                                             19980729
     US 6160088
                       Α
                                            CA 1999-2337692 19990728
     CA 2337692
                       AA
                             20000210
                                            AU 1999-53245
                                                            19990728
     AU 9953245
                       A1
                             20000221
                             20010523
                                            EP 1999-938851
                                                             19990728
     EP 1100906
                       Α1
            AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO
PRAI US 1998-124671
                       Α
                             19980729
     WO 1999-US17147
                       W
                             19990728
     Inhibitors of the KDEL receptor that can be used to block the transfer of
AΒ
     heat shock proteins to the endoplasmic reticulum and allow them to act as
     adjuvants are described. Certain proteins are functionally retained in
     the cellular endoplasmic reticulum via an interaction between a KDEL
     sequence and its receptor. According to the invention, blocking this
     interaction with a KDEL receptor inhibitor promotes the secretion of such
     proteins. In specific embodiments of the invention, KDEL receptor
     inhibitors may be used to promote the secretion of heat shock proteins,
     thereby rendering the secreted heat shock proteins more accessible to the
     immune system and improving the immune response to heat shock
     protein-assocd. antigens. The inhibitors are artificial peptides that
     oligomerize and present large no. of KDEL peptides to the receptors and
     sat. them. An example of one of these peptides uses the signal peptide of
     the BiP protein, an oligomerization domain of a cartilage oligomeric
     matrix protein, a linker peptide from a camel Ig and a KDEL peptide is
     described.
              THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE.CNT 2
              ALL CITATIONS AVAILABLE IN THE RE FORMAT
L5
      ANSWER 7 OF 9 DGENE COPYRIGHT 2003 THOMSON DERWENT ON STN
AN
      AAV41811 DNA
                          DGENE
TI
      New modified pro-domain of carboxy-peptidase B - enhances expression of
      co-expressed proteins for production of recombinant carboxy-peptidase or
      its fusions with antibodies, used, e.g. in enzyme prodrug therapy
IN
      Edge M D
PΑ
                  ZENECA LTD.
      (ZENE)
PΙ
      WO 9835988
                    A1 19980820
                                                83p
AΙ
      WO 1998-GB415
                       19980210
PRAI
      GB 1997-22727
                       19971029
      GB 1997-3104
                       19970214
      GB 1997-22003
                       19971018
DT
      Patent
LA
      English
      1998-467168 [40]
OS
DESC
      Human carboxypeptidase B pro-KDEL primer.
AΒ
      The pro-KDEL primer was used in the expression of human pancreatic
      carboxypeptidase B (CPB) from COS cells by co-secretion of pro-
      KDEL. The co-expression of a modified pro-domain of CPB from a
      separate gene enhances recombinant expression. This process can be used to produce recombinant CPB in eukaryotic cells, or fusions of CPB with
      antibody chains. CPB is used in insulin production and protein
```

sequencing, while its fusions with antibody are useful in

antibody-directed enzyme prodrug therapy. The Modified pro-domain provide increased yields of recombinant CPB, possibly by protecting the C-terminus against enzymatic degradation or by increasing intracellular trafficking.

```
L5
     ANSWER 8 OF 9 USPATFULL on STN
AN
       2000:168135 USPATFULL
       KDEL receptor inhibitors
ΤI
       Rothman, James E., New York, NY, United States
TN
       Mayhew, Mark, Tarrytown, NY, United States
       Hoe, Mee H., Irvington, NY, United States
       Sloan-Kettering Institute For Cancer, New York, NY, United States (U.S.
PA
       corporation)
PΤ
       US 6160088
                               20001212
       US 1998-124671
                               19980729 (9)
ΑI
DТ
       Utility
FS
       Granted
       Primary Examiner: Achutamurthy, Ponnathapu; Assistant Examiner: Tung,
EXNAM
       Peter P.
       Number of Claims: 13
CLMN
       Exemplary Claim: 1
ECL
DRWN
       10 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 1537
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       The present invention relates to inhibitors of the KDEL receptor and
ΔR
       therapeutic uses therefor. Certain proteins are functionally retained in
       the cellular endoplasmic reticulum via an interaction between a KDEL
       sequence and its receptor. According to the invention, blocking this
       interaction with a KDEL receptor inhibitor promotes the secretion of
       such proteins. In specific embodiments of the invention, KDEL receptor
       inhibitors may be used to promote the secretion of heat shock proteins,
       thereby rendering the secreted heat shock proteins more accessible to
       the immune system and improving the immune response to heat shock
       protein-associated antigens.
L5
     ANSWER 9 OF 9 USPATFULL on STN
AN
       1999:141293 USPATFULL
TI
       Recombinant vectors for reconstitution of liver
IN
       Kay, Mark A., Seattle, WA, United States
       Lieber, Andre, Seattle, WA, United States
PA
       University of Washington, Seattle, WA, United States (U.S. corporation)
PΙ
       US 5980886
                               19991109
       US 1997-819377
                               19970317 (8)
AΤ
RLI
       Continuation of Ser. No. US 1995-476257, filed on 7 Jun 1995, now
       abandoned which is a continuation-in-part of Ser. No. US 1994-357508,
       filed on 14 Dec 1994, now abandoned
DT
       Utility
FS
       Granted
EXNAM
       Primary Examiner: Stanton, Brian R.; Assistant Examiner: Clark, Deborah
       J. R.
       Campbell & Flores
LREP
       Number of Claims: 13
CLMN
       Exemplary Claim: 1,8
ECL
DRWN
       18 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1301
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB
       A combination of retroviral and adenoviral vectors are used for high
       efficiency gene transfer into hepatocytes, resulting in long term gene
       expression. Hepatocytes are transduced in vivo with a recombinant
       adenovirus vector that expresses a molecule capable of inducing
       hepatocyte regeneration, such as urokinase plasminogen activator (uPA)
       or tissue plasminogen activator (tPA), resulting in a high rate of liver
       regeneration. During the regenerative phase, ex vivo or in vivo
```

retroviral-mediated gene transfer into hepatocytes results in greater

transduction efficiencies. The compositions and methods thus provide new means for gene therapy, and transgenic non-human animals useful in developing new therapeutic and preventative agents.

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=>
<-----User Break----->
=> s (antigen) (3A) (release or secretion or released or secreted or release or
secrete)
  19 FILES SEARCHED...
  33 FILES SEARCHED...
  51 FILES SEARCHED...
  66 FILES SEARCHED...
         35952 (ANTIGEN) (3A) (RELEASE OR SECRETION OR RELEASED OR SECRETED OR
               RELEASE OR SECRETE)
=> (kdel) (3A) (release or secretion or released or secreted or release or secrete)
(KDEL) IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).
=> s (kdel) (3A) (release or secretion or released or secreted or secrete)
  27 FILES SEARCHED...
  54 FILES SEARCHED...
  84 FILES SEARCHED...
L7
            58 (KDEL) (3A) (RELEASE OR SECRETION OR RELEASED OR SECRETED OR
               SECRETE)
=> s 16 and 17
  63 FILES SEARCHED...
 75% OF LIMIT FOR L#S REACHED
  93 FILES SEARCHED...
             1 L6 AND L7
=> d 18 bib ab
     ANSWER 1 OF 1 USPATFULL on STN
L8
AN
       1999:40194 USPATFULL
TТ
       Method of producing single-chain Fv molecules
IN
       Jost, Carolina R., Washington, DC, United States
       Segal, David M., Rockville, MD, United States
       Huston, James S., Chestnut Hill, MA, United States
PA
       The United States of America as represented by the Department of Health
       and Human Services, Washington, DC, United States (U.S. government)
PΤ
       US 5888773
                               19990330
       US 1994-292124
AΙ
                               19940817 (8)
DT
       Utility
FS
       Granted
EXNAM
      Primary Examiner: Huff, Sheela; Assistant Examiner: Reeves, Julie E.
LREP
       Townsend and Townsend and Crew LLP
CLMN
       Number of Claims: 14
ECL.
       Exemplary Claim: 1
       12 Drawing Figure(s); 12 Drawing Page(s)
DRWN
LN.CNT 1407
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AΒ
       The invention relates to a method of producing single-chain Fv molecules
       in eukaryotic cells, and to secretable sFv proteins having at least one
       non-naturally occurring glycosylation site. The single-chain Fv
      molecules produced by this method are biologically active and capable of
       being secreted from eukaryotic cells into the cell culture medium.
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=> s 16 and (kdel) 38 FILES SEARCHED... 94 FILES SEARCHED... 26 L6 AND (KDEL) => duplicate ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove ENTER L# LIST OR (END):19 DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE, DRUGLAUNCH, DRUGMONOG2, DRUGUPDATES, FEDRIP, FOREGE, GENBANK, KOSMET, MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, RDISCLOSURE, SYNTHLINE, CHEMLIST, HSDB, MSDS-CCOHS, MSDS-OHS, RTECS, CONF, IMSDRUGCONF, DIOGENES, INVESTEXT, USAN, FORIS, FORKAT, UFORDAT, AQUIRE'. ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE DUPLICATE PREFERENCE IS 'BIOTECHABS, CAPLUS, USPATFULL, WPINDEX' KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n PROCESSING COMPLETED FOR L9 25 DUPLICATE REMOVE L9 (1 DUPLICATE REMOVED) L10 => s 110 NOT (genebank or genbank) 22 FILES SEARCHED... 95% OF LIMIT FOR L#S REACHED 42 FILES SEARCHED... 58 FILES SEARCHED... MAXIMUM L# REACHED SAVE QUERIES AND/OR ANSWER SETS IF DESIRED USE DELETE HISTORY TO DELETE ALL L'S MAXIMUM L# WOULD BE EXCEEDED SAVE QUERIES AND/OR ANSWER SETS IF DESIRED USE DELETE HISTORY TO DELETE ALL L#S If this command were executed now, it would create an L-number higher

than the maximum allowed L-number (L999). To perform this command, you must delete some or all L-numbers. SAVE any L-number queries or

answer sets you wish to retain, then execute DELETE HISTORY.

=> delete history DELETE ALL L# ITEMS? (Y)/N:y ALL L# ITEMS DELETED => s ((antigen) (3A) (release or secretion or released or secreted or release or secrete)) and (kdel) 19 FILES SEARCHED... 34 FILES SEARCHED... 54 FILES SEARCHED... 72 FILES SEARCHED... 1.1 26 ((ANTIGEN) (3A) (RELEASE OR SECRETION OR RELEASED OR SECRETED OR RELEASE OR SECRETE)) AND (KDEL) => duplicate ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove ENTER L# LIST OR (END):11 DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE, DRUGLAUNCH, DRUGMONOG2, DRUGUPDATES, FEDRIP, FOREGE, GENBANK, KOSMET, MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, RDISCLOSURE, SYNTHLINE, CHEMLIST, HSDB, MSDS-CCOHS, MSDS-OHS, RTECS, CONF, IMSDRUGCONF, DIOGENES, INVESTEXT, USAN, FORIS, FORKAT, UFORDAT, AQUIRE'. ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE DUPLICATE PREFERENCE IS 'BIOTECHABS, CAPLUS, USPATFULL, WPINDEX' KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n PROCESSING COMPLETED FOR L1 L2 25 DUPLICATE REMOVE L1 (1 DUPLICATE REMOVED) => s 12 NOT (genbank) or genebank) UNMATCHED RIGHT PARENTHESIS 'GENEBANK) ' The number of right parentheses in a query must be equal to the number of left parentheses. => s 12 NOT (genbank or genebank) 29 FILES SEARCHED... 45 FILES SEARCHED... 58 FILES SEARCHED... 92 FILES SEARCHED... T.3 17 L2 NOT (GENBANK OR GENEBANK) => d 13 1-17 bib ab ANSWER 1 OF 17 BIOTECHABS COPYRIGHT 2003 THOMSON DERWENT/ISI on STN 1.3 AN 2002-17633 BIOTECHABS ΤI Preparing recombinant vector containing reporter and therapeutic genes, useful for treatment of fibrosis, particularly of liver, by inducing degradation of collagen; recombinant adeno virus production expressing beta-galactosidase, human urokinase, transforming growth factor and growth factor gene ΑU ARMENDARIZ BORUNDA J; AGUILAR CORDOVA E PA TGT LAB SA DE CV WO 2002044393 6 Jun 2002 PΙ AΙ WO 2000-MX50 28 Nov 2000 PRAI MX 2000-1713 28 Nov 2000 DT Patent LA Spanish os WPI: 2002-471834 [50] AB DERWENT ABSTRACT: NOVELTY - Preparing recombinant (non-)viral vectors (A) by cloning reporter gene (RG), and modified cDNA (I) of a therapeutic gene (II) that encodes a protein (III) useful for treating fibrosis (hepatic, pulmonary, renal, cardiac or pancreatic), keloids and hypertrophic scars in mammals, including humans, is new. DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a

recombinant adenoviral vector (A') produced by the new method. BIOTECHNOLOGY - Preferred Materials: RG is a bacterial beta-galactosidase (lacZ) gene. (III) is: (a) optionally modified human urokinase-type plasminogen activator (huPA) (this activates latent hepatic collagenases and matrix metalloproteases, and restores replication of hepatocytes); (b) truncated type II TGF (transforming growth factor) beta receptor (this degrades and/or prevents synthesis/deposition of collagen proteins in cirrhotic organs); or (c) hepatic growth factor (this induces hepatic regeneration, leading to an increase in the number of hepatocytes positive for cellular proliferation nuclear antigen). A non-secreted form of huPA is expressed, i.e. one that has a signal for retention in the endoplasmic reticulum (ER), specifically KDEL, at the C-terminus, also, at the N-terminus, a sequence that encodes a retention signal linked to a transmembrane region anchor. Preferred Vectors: The vectors are adenoviral (1st-4th generation or 'gutless'), retroviral or adeno-associated viral vectors, or plasmids or cationic and anionic liposomes. Especially, preferred is a recombinant adenoviral vector in which the E1 and/or E3 open reading frames have been removed (but leaving sufficient sequence for vector replication in vitro) and the inserted gene or cDNA is under control of an ubiquitous, tissue-specific and/or inducible or regulatable promoter, especially the cytomegalovirus promoter. Preferred Process: Expression of the therapeutic gene can be monitored by measuring expression of the corresponding human protein (enzyme-linked immunosorbent assay or immunohistochemical methods), and expression of endogenous collagen genes may be followed by semi-quantitative reverse-transciption polymerase chain reaction.

ACTIVITY - Hepatotropic; Antifibrotic; Vulnerary.

MECHANISM OF ACTION - (II) induce degradation of collagen. Liver cirrhosis was induced in rats using tetrachloromethane, then the animals injected (iliac vein) with a single dose of 6x10 to the power 11 viral particles/kg of vector pAd.PGk-DELTANDELTAC-huPA (expressing human urokinase). Analysis of the liver after 8 days indicated levels (international units/l) of ALT, AST and alkaline phosphatase of 88.5, 137 and 205; compare 410, 2250 and 454 for animals injected with an irrelevant vector; and total bilirubin was 0.94 (compare 1.4) mg/dl. Hematological parameters were not affected and the treatment significantly increased expression of matrix metalloprotease-2; reduced expression of collagens types I, III and IV, and stimulated mitosis of hepatocytes.

USE - (A) are used to treat fibrosis in the cirrhotic liver, but more generally fibrosis in any organ.

ADMINISTRATION - Targeting to fibrotic organs is achieved by selecting the route of administration (specifically intravenous); from the natural tropism (to liver) of the vector, or by vector selection. The unit dose is 107-1014 viral particles.

ADVANTAGE - (A) do not secrete significant amounts of plasminogen activator, so cause neither hypocoagulation nor spontaneous bleeding.

EXAMPLE - The cDNA for human urokinase plasminogen activator (huPA) was cloned into the XhaI/Asp718 sites of pGEM3 and modified by attachment of a sequence encoding the signal KDEL, for retention in the endoplasmic reticulum, at the C-terminus and a 75 base pair polymerase chain reaction fragment to replace the pre-uPA signal sequence with a retention signal and transmembrane anchor from the transmembrane protein IiP33. The modified cDNA was then cloned (no details) to form vector pAdPGK-DELTANDELTAC-huPA, for subsequent production of recombinant adenoviral particles. (74 pages)

L3 ANSWER 2 OF 17 CAPLUS COPYRIGHT 2003 ACS ON STN AN 1993:58075 CAPLUS

DN 118:58075

TI The calcium-binding protein calreticulin is a major constituent of lytic granules in cytolytic T lymphocytes

AU Dupuis, Marc; Schaerer, Esther; Krause, Karl Heinz; Tschopp, Juerg

CS Inst. Biochem., Univ. Lausanne, Epalinges, CH-1066, Switz.

SO Journal of Experimental Medicine (1993), 177(1), 1-7

CODEN: JEMEAV; ISSN: 0022-1007

DT Journal

LA English

AB Cytolytic T lymphocytes (CTL), natural killer cells, and lymphokine-activated killer (LAK) cells are cytolytic cell release the cytolytic protein perforin and a family of progranzymes, from cytoplasmic stores upon interaction with the part the parties of an addal major 60

lymphokine-activated killer (LAK) cells are cytolytic cells known to release the cytolytic protein perforin and a family of proteases, named granzymes, from cytoplasmic stores upon interaction with target cells. Here, the authors report the purifn. of an addnl. major 60-kD granule-assocd. protein (grp 60) from human LAK cells and from mouse cytolytic T cells. The N-terminal amino acid sequence of the polypeptide was found to be identical to calreticulin. Calreticulin is a Ca storage protein and carries a C-terminal KDEL sequence, known to act as a retention signal for proteins destined to the lumen of the endoplasmic reticulum. In CTLs, however, calreticulin colocalizes with the lytic perforin to the lysosome-like secretory granules, as confirmed by double label immunofluorescence confocal microscopy. Moreover, when the release of granule-assocd. proteins was triggered by stimulation of the T cell receptor complex, calreticulin was released along with granzymes A and D. Since perforin is activated and becomes lytic in the presence of Ca, it is proposed that the role of calreticulin is to prevent organelle autolysis. due to the protein's Ca chelator capacity.

1.3 ANSWER 3 OF 17 USPATFULL on STN AN 2003:232060 USPATFULL ΤI Vaccine adjuvant IN Minion, F. Chris, Ames, IA, UNITED STATES Menon, Sreekumar A., Philadelphia, PA, UNITED STATES Mahairas, Gregory G., Seattle, WA, UNITED STATES PΑ Iowa State University Research Foundation, Inc., an Iowa corporation (U.S. corporation) ΡI US 2003162260 A1 20030828 ΑI US 2003-384948 Α1 20030310 (10) RLI Division of Ser. No. US 2000-692064, filed on 19 Oct 2000, GRANTED, Pat. No. US 6537552 PRAI US 1999-160249P 19991019 (60) DT Utility FS APPLICATION LREP FISH & RICHARDSON P.C., 3300 DAIN RAUSCHER PLAZA, 60 SOUTH SIXTH STREET, MINNEAPOLIS, MN, 55402 Number of Claims: 7 CLMN ECL Exemplary Claim: 1 7 Drawing Page(s) DRWN LN.CNT 1632 The invention features fusion agents such as fusion proteins that are useful for the treatment of and prevention from diseases that are susceptible to the effects of cellular (Th1 type) immune responses. Also encompassed by the invention are nucleic acids encoding the fusion proteins of the invention, vectors containing the nucleic acids, and cells containing the vectors. The invention includes methods of making and using the fusion agents of the invention.

L3 ANSWER 4 OF 17 USPATFULL on STN ΑN 2003:165491 USPATFULL Hybrid lt-a/ct-b holotoxin for use as an adjuvant ΤI IN Clements, John D, New Orleans, LA, UNITED STATES PΙ US 2003113345 **A**1 20030619 ΑI US 2002-276844 A1 20021119 (10) WO 2001-US16542 20010521 DT Utility FS APPLICATION LREP PENNIE AND EDMONDS, 1155 AVENUE OF THE AMERICAS, NEW YORK, NY, 100362711 CLMN Number of Claims: 22

Exemplary Claim: 1 ECL DRWN 6 Drawing Page(s) LN.CNT 1259 CAS INDEXING IS AVAILABLE FOR THIS PATENT. The present invention provides a novel composition which is a hybrid AB heat labile enterotoxin comprising the A-subunit of the heat labile toxin of Escherichia coli (LT-A) and the B-subunit of the cholera enterotoxin of Vibrio cholerae (CT-B). The hybrid toxin is designated LT-A/CT-B. The LT-A subunit, the CT-B subunit, or both subunits of the hybrid toxin may be mutant subunits, e.g., differing from wild-type subunits by amino acid substitutions, deletions or additions. Also provided are methods of using the novel LT-A/CT-B comprising compositions of the invention as adjuvants for vaccines, methods of making the LT-A/CT-B hybrid holotoxin, and kits. L3ANSWER 5 OF 17 USPATFULL on STN ΑN 2003:81455 USPATFULL TI Vaccine adjuvant IN Minion, F. Chris, Ames, IA, United States Menon, Sreekumar A., Philadelphia, PA, United States Mahairas, Gregory G., Seattle, WA, United States PA Iowa State University Research Foundation, Ames, IA, United States (U.S. corporation) PΙ US 6537552 B1 20030325 US 2000-692064 ΑI 20001019 (9) PRAI US 1999-160429P 19991019 (60) DT Utility FS GRANTED EXNAM Primary Examiner: Smith, Lynette R. F.; Assistant Examiner: Shahnan-Shah, Khatol S LREP Fish & Richardson P.C. CLMN Number of Claims: 8 ECL Exemplary Claim: 1 DRWN 10 Drawing Figure(s); 7 Drawing Page(s) LN.CNT 1611 CAS INDEXING IS AVAILABLE FOR THIS PATENT. AΒ The invention features fusion agents such as fusion proteins that are useful for the treatment of and prevention from diseases that are susceptible to the effects of cellular (Th1 type) immune responses. Also encompassed by the invention are nucleic acids encoding the fusion proteins of the invention, vectors containing the nucleic acids, and cells containing the vectors. The invention includes methods of making and using the fusion agents of the invention. ANSWER 6 OF 17 USPATFULL on STN L32003:40768 USPATFULL AN TI Pharmaceuticals for modulating hormone responsiveness TN Dedhar, Shoukat, #1-219 East 8th St., North Vancouver B.C., CANADA V7L 1Y9 рT US 6518397 В1 20030211 ΑТ US 1997-900241 19970724 (8) RLI Continuation-in-part of Ser. No. US 377432, now patented, Pat. No. US 5854202 DТ Utility FS GRANTED EXNAM Primary Examiner: Eyler, Yvonne; Assistant Examiner: Brannock, Michael LREP Burns, Doane, Swecker & Mathis, L.L.P. CLMN Number of Claims: 4 ECL Exemplary Claim: 1 DRWN 12 Drawing Figure(s); 11 Drawing Page(s) LN.CNT 1930 CAS INDEXING IS AVAILABLE FOR THIS PATENT. AΒ This invention relates to isolated and purified proteins, such as

calreticulin and mimetics and inhibitors of calreticulin, for a novel use of modulating hormone responsiveness. These proteins are useful in gene therapy and in manufacturing pharmaceuticals for treating a variety of diseases, including cancer, osteoporosis and chronic inflammatory disease. The proteins include or bind to an amino acid sequence [SEQ ID NO: 1] KXFFX.sup.1R, wherein X is either G, A or V and Y is either K or R. This sequence is present in the DNA-binding domain, and is critical for the DNA binding activity, of a variety of hormone receptors, including glucocorticoid receptor, minerolcorticoid receptor, androgen receptor, progesterone receptor, estrogen receptor, retinoic acid receptor, thyroid hormone receptor and vitamin D receptor. Proteins which bind to this sequence may inhibit hormone receptor induced gene transcription. Proteins which include this sequence may promote hormone receptor induced gene transcription.

ANSWER 7 OF 17 USPATFULL on STN

L3 AN

```
2002:258433 USPATFULL
TΙ
       Anti-CD3 immunotoxins and therapeutic uses therefor
TN
       Digan, Mary Ellen, Morristown, NJ, UNITED STATES
       Lake, Philip, Morris Plains, NJ, UNITED STATES
       Wright, Richard Michael, Annandale, NJ, UNITED STATES
PΙ
       US 2002142000
                          Αl
                               20021003
ΑI
       US 2000-480236
                          Α1
                               20000110 (9)
DТ
       Utility
FS
       APPLICATION
LREP
       THOMAS HOXIE, NOVARTIS CORPORATION, PATENT AND TRADEMARK DEPT, 564
       MORRIS AVENUE, SUMMIT, NJ, 079011027
CLMN
       Number of Claims: 29
ECL
       Exemplary Claim: 1
       23 Drawing Page(s)
DRWN
LN.CNT 2935
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Recombinant immunotoxin polypeptides are described comprising a
AB
       CD3-binding domain and a Pseudomonas exotoxin mutant, and in particular,
       comprising a single chain (sc) Fv as the CD3-binding moiety. A preferred
       species of the invention comprises scFv(UCHT-1)-PE38. Also disclosed are
       methods for the preparation of said immunotoxins; functionally
       equivalent immunotoxins which are intermediates in the preparation of
       the immunotoxins of the invention, as well as polynucleotide and
       oligonucleotide intermediates; methods for the prevention and/or
       treatment of transplant rejection and induction of tolerance, as well as
       treatment of autoimmune and other immune disorders, using the
       immunotoxins or pharmaceutically acceptable salts thereof; and
       pharmaceutical compositions comprising the immunotoxins or
       pharmaceutically acceptable salts thereof.
L3
     ANSWER 8 OF 17 USPATFULL on STN
AN
       2002:198276 USPATFULL
ΤI
       IMPROVEMENTS IN OR RELATING TO PEPTIDE DELIVERY
IN
       CARDY, DONALD LEONARD NICHOLAS, NORTHAMPTONSHIRE, UNITED KINGDOM
       CARR, FRANK JOSEPH, BALMEDIE, UNITED KINGDOM
                          A1
PΙ
       US 2002106370
                               20020808
AΙ
       US 1997-737457
                          A1
                               19970312 (8)
       WO 1995-GB1107
                               19950515
PRAI
       GB 1994-9643
                           19940513
       GB 1994-17461
                           19940831
DT
       Utility
FS
       APPLICATION
LREP
       ORRIN M HAUGEN, HAUGEN AND NIKOLAI, 820 INTERNATIONAL CENTRE, 900 SECOND
       AVENUE SOUTH, MINNEAPOLIS, MN, 554023325
CLMN
       Number of Claims: 23
ECL
       Exemplary Claim: 1
       10 Drawing Page(s)
DRWN
LN.CNT 751
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CAS INDEXING IS AVAILABLE FOR THIS PATENT. Disclosed is a chimaeric polypeptide comprising: a binding portion AB having specific binding affinity for a eukaryotic target cell surface component and an effector portion comprising an amino acid sequence capable of exerting a biological effect; whereby binding of the polypeptide to the cell surface component induces internalization of at least the effector portion so as to allow the amino acid sequence to exert its biological effect, together with a vaccine comprising the chimaeric polypeptide of the invention, and a method of modulating the immune response of a human or animal subject. ANSWER 9 OF 17 USPATFULL on STN L3 AN 2002:188125 USPATFULL ΤТ Protease-activatable pseudomonas exotoxin A-like proproteins TN Fitzgerald, David J., Rockville, MD, United States Reiter, Yoram, Ness Ziona, ISRAEL Pastan, Ira, Potomac, MD, United States PΑ The United States of America as represented by the Secretary of the Department of Health and Human Services, Washington, DC, United States (U.S. government) PΙ 20020730 US 6426075 В1 WO 9820135 19980514 US 1999-297851 AΙ 19990730 (9) WO 1997-US20207 19971105 19990730 PCT 371 date PRAI US 1996-30376P 19961106 (60) Utility DТ FS GRANTED EXNAM Primary Examiner: Navarro, Mark; Assistant Examiner: Baskar, Padma LREP Townsend and Townsend and Crew, LLP Number of Claims: 19 CLMN ECL Exemplary Claim: 1 DRWN 9 Drawing Figure(s); 11 Drawing Page(s) LN.CNT 2738 CAS INDEXING IS AVAILABLE FOR THIS PATENT. This invention provides protease-activatable Pseudomonas exotoxin A-like AΒ ("PE-like") proproteins. The proproteins comprise (1) a cell recognition domain of between 10 and 1500 amino acids that binds to a cell surface receptor; (2) a modified PE translocation domain comprising an amino acid sequence sufficiently homologous to domain II of PE to effect translocation to a cell cytosol upon proteolytic cleavage, wherein the translocation domain comprises a cysteine-cysteine loop that comprises a protease activatable sequence cleavable by a protease and wherein the cysteine-cysteine loop is substantially un-activatable by furin; (3) optionally, a PE Ib-like domain comprising an amino acid sequence up to 1500 amino acids; (4) a cytotoxicity domain comprising an amino acid sequence substantially homologous to domain III of PE, the cytotoxicity domain having ADP-ribosylating activity; and (5) an endoplasmic reticulum ("ER") retention sequence. The invention also provides methods of using these proproteins for killing target cells. ANSWER (10) OF 17 USPATFULL on STN L3 AN 2002:181537 USPATFULL TI Polynucleotides encoding protease-activatable pseudomonas exotoxin a-like proproteins IN Fitzgerald, David J., Rockville, MD, United States Reiter, Yoram, Ness Ziona, ISRAEL Pastan, Ira, Potomac, MD, United States PAThe United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government) PΙ US 6423513 B1 20020723 AΤ US 2000-479479 20000110 (9) RLI Division of Ser. No. US 297851 PRAI US 1996-30376P 19961106 (60)

DTUtility FS GRANTED Primary Examiner: Navarro, Mark; Assistant Examiner: Baskar, Padmavathi EXNAM Townsend and Townsend and Crew, LLP LREP Number of Claims: 5 CLMN Exemplary Claim: 1 ECL 11 Drawing Figure(s); 11 Drawing Page(s) DRWN LN.CNT 2665 CAS INDEXING IS AVAILABLE FOR THIS PATENT. This invention provides protease-activatable Pseudomonas exotoxin A-like AB ("PE-like") proproteins. The proproteins comprise (1) a cell recognition domain of between 10 and 1500 amino acids that binds to a cell surface receptor; (2) a modified PE translocation domain comprising an amino acid sequence sufficiently homologous to domain II of PE to effect translocation to a cell cytosol upon proteolytic cleavage, wherein the translocation domain comprises a cysteine-cysteine loop that comprises a protease activatable sequence cleavable by a protease and wherein the cysteine-cysteine loop is substantially un-activatable by furin; (3) optionally, a PE Ib-like domain comprising an amino acid sequence up to 1500 amino acids; (4) a cytotoxicity domain comprising an amino acid sequence substantially homologous to domain m of PE, the cytotoxicity domain having ADP-ribosylating activity; and (5) an endoplasmic reticulum ("ER") retention sequence. The invention also provides methods of using these proproteins for killing target cells. ANSWER 11 OF 17 USPATFULL on STN L3AN2002:1088 USPATFULL Recombinant Haemophilus influenzae adhesin proteins TT TN Loosmore, Sheena M., Aurora, CANADA Yang, Yan Ping, Willowdale, CANADA Klein, Michel H., Willowdale, CANADA Aventis Pasteur Limited, Toronto, CANADA (non-U.S. corporation) PA PΤ 20020101 US 6335182 В1 ΑI US 1999-268347 19990316 (9) DТ Utility FS GRANTED Primary Examiner: Graser, Jennifer E. EXNAM LREP Sim & McBurney CLMN Number of Claims: 24 ECL Exemplary Claim: 1 DRWN 206 Drawing Figure(s); 201 Drawing Page(s) LN.CNT 2173 CAS INDEXING IS AVAILABLE FOR THIS PATENT. Recombinant production of Hia protein, in full-length and N-terminally truncated forms, of non-typeable strains of Haemophilus influenzae, is described. The nucleic acid and deduced amino acid sequences of Hia genes of various strains of non-typeable and type c Haemophilus influenzae also are described. ANSWER 12 OF 17 USPATFULL on STN L3 2001:93332 USPATFULL AN Immunization with plasmid encoding immunogenic proteins and ΤI intracellular targeting sequences IN Williams, William V., Havertown, PA, United States Madaio, Michael, Bryn Mawr, PA, United States Weiner, David B., Merion Station, PA, United States The Trustees of the University of Pennsylvania, Philadelphia, PA, United PA States (U.S. corporation) PΤ US 6248565 В1 20010619 ДΤ US 2000-496301 20000202 (9) Continuation of Ser. No. US 1997-957001, filed on 23 Oct 1997 RLIPRAI US 1996-29592P 19961023 (60) DT Utility FS GRANTED

Primary Examiner: Park, Hankyel T. EXNAM LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP CLMN Number of Claims: 35 ECL Exemplary Claim: 1 22 Drawing Figure(s); 14 Drawing Page(s) DRWN LN.CNT 1952 CAS INDEXING IS AVAILABLE FOR THIS PATENT. Improved vaccines are disclosed. The improved vaccines include a AB nucleotide sequence that encodes a coding sequence that comprises an immunogenic target protein linked to or comprising an intracellular cellular targeting sequence, the coding sequence being operably linked to regulatory elements are disclosed. Methods of immunizing individuals are disclosed. ANSWER 13 OF 17 USPATFULL on STN L3 AN2001:67432 USPATFULL TТ Plasmids encoding immunogenic proteins and intracellular targeting sequences IN Williams, William V., Havertown, PA, United States Madaio, Michael, Bryn Mawr, PA, United States Weiner, David B., Merion Station, PA, United States The Trustees of the University of Pennsylvania, Philadelphia, PA, United PA States (U.S. corporation) PΙ US 6228621 В1 20010508 US 1997-957001 AΙ 19971023 (8) US 1996-29592P PRAI 19961023 (60) DTUtility FS Granted EXNAM Primary Examiner: Park, Hankyel LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP CLMN Number of Claims: 40 Exemplary Claim: 1 ECL 22 Drawing Figure(s); 14 Drawing Page(s) DRWN LN.CNT 1897 CAS INDEXING IS AVAILABLE FOR THIS PATENT. Improved vaccines are disclosed. The improved vaccines include a nucleotide sequence that encodes a coding sequence that comprises an immunogenic target protein linked to or comprising an intracellular cellular targeting sequence, the coding sequence being operably linked to regulatory elements are disclosed. Methods of immunizing individuals are disclosed. ANSWER 14 OF 17 USPATFULL on STN L31999:40194 USPATFULL AN TT Method of producing single-chain Fv molecules IN Jost, Carolina R., Washington, DC, United States Segal, David M., Rockville, MD, United States Huston, James S., Chestnut Hill, MA, United States PA The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government) PΤ US 5888773 19990330 US 1994-292124 AΙ 19940817 (8) DТ Utility FS Granted Primary Examiner: Huff, Sheela; Assistant Examiner: Reeves, Julie E. EXNAM Townsend and Townsend and Crew LLP LREP Number of Claims: 14 CLMN ECL Exemplary Claim: 1 DRWN 12 Drawing Figure(s); 12 Drawing Page(s) LN.CNT 1407 CAS INDEXING IS AVAILABLE FOR THIS PATENT. AR The invention relates to a method of producing single-chain Fv molecules in eukaryotic cells, and to secretable sFv proteins having at least one

non-naturally occurring glycosylation site. The single-chain Fv

molecules produced by this method are biologically active and capable of being secreted from eukaryotic cells into the cell culture medium.

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ANSWER 15 OF 17 USPATFULL on STN
L3
       1998:161992 USPATFULL
AN
       Genetically engineered chimeric viruses for the treatment of diseases
ΤI
       associated with viral transactivators
       Tattersall, Peter J., Guilford, CT, United States
IN
       Cotmore, Susan F., Guilford, CT, United States
       Yale University, New Haven, CT, United States (U.S. corporation)
PA
       US 5853716
                               19981229
PΙ
       US 1996-690174
                               19960725 (8)
ΑI
       US 1995-1611P
                           19950728 (60)
PRAI
       Utility
DТ
       Granted
FS
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Yucel, Irem
LREP
       Pennie & Edmonds LLP
       Number of Claims: 12
CLMN
ECL
       Exemplary Claim: 1
       16 Drawing Figure(s); 12 Drawing Page(s)
DRWN
LN.CNT 2322
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       The present invention relates to chimeric viruses, the replication of
AB
       which is regulated by a transactivation signal produced by diseased host
       cells. The chimeric viruses of the invention can infect both normal and
       diseased host cells. However, the chimeric virus replicates efficiently
       in and kills diseased host cells that produce the transactivation
       signal. The use of such chimeric viruses to treat infectious diseases
       and cancers are described. A particularly useful embodiment involves the
       modification of a murine parvovirus that infects human T cells to
       generate a chimeric parvovirus that is cytocidal to human T cells that
       express HIV-tat. The chimeric parvovirus can be used to treat
       HIV-infection.
     ANSWER 16 OF 17 USPATFULL on STN
L3
AN
       96:11071 USPATFULL
ΤI
       Monoclonal antibodies to prostate cells
       Pastan, Ira H., Potomac, MD, United States
TN
       The United States of America as represented by the Department of Health
PA
       and Human Services, Washington, DC, United States (U.S. government)
PТ
       US 5489525
                               19960206
       US 1992-958140
AΙ
                               19921008 (7)
DT
       Utility
       Granted
FS
      Primary Examiner: Scheiner, Toni R.
EXNAM
       Townsend and Townsend Khourie and Crew
LREP
       Number of Claims: 11
CLMN
       Exemplary Claim: 7
ECL
       13 Drawing Figure(s); 6 Drawing Page(s)
DRWN
LN.CNT 1450
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB
       Monoclonal antibodies are provided which bind to an antigen associated
       with prostate cells, including prostate cancers. The monoclonal
       antibodies and recombinant forms thereof are used individually or
       conjugated radioisotopes to target the compounds to cancerous prostate
       cells, and thus are useful in a variety of diagnostic procedures.
     ANSWER 17 OF 17 WPINDEX COPYRIGHT 2003 THOMSON DERWENT on STN
L3
     1999-562102 [47]
                        WPINDEX
ΑN
DNC
    C1999-163981
     New polynucleotides encoding antigens which are presented with MHC Class I
     and II molecules, used for treating e.g. tumors, infections, autoimmune
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DC B04 D16

disorders, allergies or allograft rejection.

ROBERTS, B L IN PΑ (GENZ) GENZYME CORP CYC 23 PΙ WO 9947641 A1 19990923 (199947)* EN 82p RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE W: AU CA JP US AU 9931022 A 19991011 (200008) EP 1064354 A1 20010103 (200102) EN R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE JP 2002506633 W 20020305 (200220) 94p ADT WO 9947641 A1 WO 1999-US6030 19990319; AU 9931022 A AU 1999-31022 19990319; EP 1064354 A1 EP 1999-912709 19990319, WO 1999-US6030 19990319; JP 2002506633 W WO 1999-US6030 19990319, JP 2000-536824 19990319 AU 9931022 A Based on WO 9947641; EP 1064354 A1 Based on WO 9947641; JP 2002506633 W Based on WO 9947641 PRAI US 1998-78725P 19980320 9947641 A UPAB: 19991116 NOVELTY - A novel polynucleotide (PN), referred to as (A), encodes an antigen that is processed and presented with a major histocompatibility complex (MHC) Class I molecule on an antigen-presenting cell (APC) and an antigen that is processed and presented with an MHC Class II molecule on the APC.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) a gene delivery vehicle comprising a PN as in (A);
- (2) a host cell comprising a PN as in (A);
- (3) a polypeptide encoded by a PN as in (A);
- (4) a method of expressing (A) by culturing the cell of (2) under standard culture conditions;
- (5) a method of increasing presentation of a peptide on the surface of an APC comprises introducing (A) into the cell under conditions which favor the expression of the polynucleotide;
 - (6) a cell produced by the method of (5);
- (7) a method of producing a population of educated, antigen-specific immune effector cells comprises culturing naive immune effector cells with an APC transduced with (A);
- (8) a population of educated, antigen-specific immune effector cells produced by the method of (7);
- (9) a method of inducing an immune response to an antigen in a subject comprises administering (A) under conditions that induce an immune response to the antigen;
- (10) a method of inducing an immune response to a native antigen in a subject comprises administering the host cell of (2) under conditions that induce an immune response to the antigen; and
- (11) a method of adoptive immunotherapy comprises administering to an individual an effective amount of the cells of (8).

ACTIVITY - Immunosuppressive; Antiallergic.

MECHANISM OF ACTION - None given.

USE - The PNs can use both MHC Class I and Class II presenting pathways, in the same APC, to modulate a humoral and a cellular immune response in a subject against a given antigen. The APCs transduced with the PNs can be used for producing a population of educated, antigen-specific immune effector cells (claimed). The PNs can be used for inducing an immune response to an antigen in a subject (claimed). The antigen may be a tumor-associated antigen, e.g. gp100, MUC-1, MART-1, HER-2, CEA, PSA, prostate specific membrane antigen, tyrosinase, tyrosinase related proteins 1 or 2, NY-ESO-1 or GA733 antigen (claimed). The PNs are useful in methods in induce, increase, or enhance an immune response to a pathogenic organism, e.g. pathogenic viruses, bacteria or protozoans. They can also be used to treat disorders such as autoimmune disorders, allergies, or allograft rejection. The PNs can also be used in diagnostic applications.

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